conserved throughout evolution. Multiple homologues to the bacterial genes *MutS* and *MutL* have been identified in organisms ranging from yeast to man. To date six *MutS* and three *MutL* homologues have been identified in S. *cerevisiae*. *PMS1* and *MSH2* are yeast homologues of the bacterial mismatch repair genes *MutL* and *MutS* respectively. A dramatic increase in the frequency of the post-meiotic segregation of genetic markers is observed in *pms1* and *msh2* mutants. This is indicative of unrepaired heteroduplex, suggesting a role for these genes in the process of gene conversion. *pms1* and *msh2* mutants also have a mutator phenotype similar to that of bacterial mismatch repair mutants. This reflects a deficiency in repair of DNA synthesis errors and spontaneous DNA lesions. Both gene products have also been shown to form part of a ternary complex that assembles *in vitro* at mismatched base-pairs in duplex DNA.

Antirecombination has profound implications for the process of meiosis. During meiotic prophase the formation of a physical connection between homologues, in the form of a crossover, allows correct orientation on the meiosis I spindle. This ensures the faithful disjunction of chromosomes to produce viable, haploid gametes. Mutations that reduce or abolish meiotic crossing over cause low spore viability, presumably due to extensive chromosomal nondisjunction. In yeast, as in bacteria, reduced chromosomal identity (~10-30% DNA sequence divergence) acts as a barrier to recombination and during meiosis, dramatically reduces exchange between homologues. The repression of recombination between homologous chromosomes during meiosis may lead to the reproductive isolation of populations in the form of sterility.

Alani *et al.*, 1994 (*Genetics* **137**: 19-39) describe interaction between mismatch repair and genetic recombination in non-homeologous situations in yeast. On the basis of an increased gene conversion frequency in msh2 mutants, the authors speculate that mismatch repair

proteins such as that coded for by msh2 might be involved in preventing homeologous recombination, but have no evidence to support this.

Prolla *et al.*, 1994 (*Molecular and Cellular Biology* **14**: 407-415). describe the identification of a new gene, MLH1, and its role in mismatch repair. The authors measured the effect of the mutants on non-homeologous simple sequence repeats and single base mismatches.

European patent specification 449 923 of Setratech is directed to a process of intergeneric recombination *in vivo* of partially homologous DNA sequences having up to 30% of base mismatches, characterized in that the sequences are placed together in cells or an